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The Use of the Abdominal Aortic and Junctional Tourniquet During Cardiopulmonary Resuscitation Following Traumatic Cardiac Arrest in Swine

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ABSTRACT Background: Standard cardiopulmonary resuscitation (CPR) is ineffective in treating traumatic cardiac arrest (TCA) following hemorrhagic shock despite fluid resuscitation. CPR adjuncts, including abdominal compressions and external counter pressure, have shown some success in laboratory settings. The Abdominal Aortic and Junctional Tourniquet (AAJT) is a device that occludes both venous and arterial blood at the level of the aortic bifurcation and likely increases thoracic pressure when applied to the abdomen. We developed a swine model of controlled hemorrhage to induce a state of TCA to test the ability of the AAJT to improve the efficacy of CPR. Methods: Twelve splenectomized, Yorkshire, male swine (70–90 kg) were randomized into two groups: presence or absence of AAJT placement. Controlled hemorrhage was performed at a rate of 2 mL/kg/min until systolic blood pressure reached below 10 mm Hg (defined as cardiac arrest). Following 3 minutes of arrest, the animals underwent CPR using a mechanical compression device along with either the presence or absence of the AAJT. Concurrently, 5 units of whole blood (2,500 mL) were delivered through the jugular vein at 500 mL/min. Efficacy of CPR was assessed by analyzing rates of return of spontaneous circulation (ROSC) and survival. Blood pressure, carotid blood flow, and other hemodynamic values were also compared. Findings: No significant differences between groups were observed before treatments. The controlled hemorrhage resulted in an average loss of 2,654 ± 323 g of blood over 18.2 ± 3.9 minutes. All animals that had a ROSC survived to the end of the 1-hour observation period. Animals with AAJT survived 83% (5/6) compared to 17% (1/6) of animals without AAJT. Finally, blood pressure, carotid flow, mean pulmonary artery pressure, and end tidal carbon dioxide were all significantly different between groups at the end of the first 10-minute compression period. Discussion/Impact/Recommendations: These results suggest that the AAJT could allow for increased CPR efficacy in cases of TCA when used in conjunction with rapid, massive blood transfusions.

INTRODUCTION

During the recent conflicts in Iraq and Afghanistan, 90% of potentially survivable deaths were associated with hemorrhage.[1] The end result of severe hemorrhage is traumatic cardiac arrest (TCA). Current treatments focus on stopping the bleeding early before TCA can occur. The widespread use of tourniquets has led to a decrease in the number of deaths due to extremity injuries.[2] However, there are not equivalent treatments for junctional or torso injuries. Junctional tourniquets, hemostatic gauzes, and resuscitative balloon occlusion of the aorta have all been developed to treat some of these injuries, but none have seen general adoption or use.[3–8] Successful approaches to treating TCA will likely result in improved prehospital care of traumatic hemorrhage.

Cardiopulmonary resuscitation (CPR) is currently believed to be ineffective in cases of TCA due to the extreme state of hypovolemia.[9–11] A previous study in our lab found that CPR with large amounts of intravenous blood is ineffective in treating TCA (unpublished results) in a model of noncompressible torso hemorrhage.[12,13] Furthermore, a recent study evaluating the utility of CPR in a canine model showed no benefit of compressions in hypovolemic animals.[14] This study showed the need for resuscitation fluids following pulseless electrical activity with no benefit of CPR. However, the 2014 Tactical Combat Casualty Care guidelines still recommend CPR when "the casualty does not have obviously fatal wounds and will be arriving at a facility with a surgical capability within a short period of time."

Many adjuncts have been tested in conjunction with standard chest compressions, yielding mixed results.[15–17] CPR with military anti-shock trousers in place showed some improvement by increased aortic systolic pressure, but no improvement in survival or organ blood flow.[17] Abdominal binders and a large pneumatic abdominal cuff also showed improvements in animal models including increased carotid blood flow, blood pressure, and even survival.[18] More recently, positive pressure ventilation and compression–decompression during CPR have shown some improvement in both animal models and clinical trials.[16,19,20] However, these CPR adjuncts have only been shown to improve CPR in non-TCA.

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The views expressed are those of the authors and do not reflect the official views of the Department of Defense or its components. The experiments reported herein were conducted according to the principles set forth in the National Institute of Health Publication No. 80-23, Guide for the Care and Use of Laboratory Animals and the Animal Welfare Act of 1996, as amended.

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The Abdominal Aortic and Junctional Tourniquet (AAJT; Compression Works, Birmingham, Alabama) is a $525 device that uses an air-filled bladder to restrict blood flow distal to the tourniquet when placed on the abdomen or junction sites. It has been studied in both laboratory setting and used in the field, but is not recommended by the committee on Tactical Combat Casualty Care due to concerns over time of application (approximately 1 minute), risk of increased proximal bleeding, and possible pulmonary compromise.[21–26] A recent study shows that some of these harmful physiologic effects could be mitigated via intubation and mechanical ventilation.[23] When placed on the abdomen, the device occludes the descending aorta and the inferior vena cava. Along with blocking blood flow, the AAJT may increase thoracic pressure due to limited diaphragmatic motion when placed on the abdomen. This increase in thoracic pressure may confine the energy from compressions to the thoracic cavity and thereby the heart.

The purpose of this study was to determine the impact of the AAJT on survival and return of spontaneous circulation (ROSC) in a TCA CPR swine model. Hemodynamic and blood chemistry were also compared between groups.

METHODS

This study was a prospective, randomized, blinded experimental trial. All procedures were performed at a facility accredited by the Association for the Assessment and Accreditation of Laboratory Animal Care. Animals were used in accordance with the *Guide for the Care and Use of Laboratory Animals*, and the protocol was approved of by the United States Air Force 59th Medical Wing's Institutional Animal Care and Use Committee.

Surgical Procedures

Male Yorkshire–Landrace swine weighing between 70 and 90 kg (John Albert Farms, Cibolo, Texas) were used in all procedures. Animals were sedated with 4.4 mg/kg tiletamine–zolazepam and 2.2 mg/kg ketamine. Buprenorphine was then given for alleviation of pain at 0.01 mg/kg. Following intubation, anesthesia was induced by up to 4% isoflurane. Once stabilized, isoflurane was dropped to maintain a minimum alveolar concentration of greater than 1.2. No maintenance fluid was administered. Animals were placed on a wedge-shaped, custom-made, surgical table in order to stabilize animals during chest compressions.

All vascular access was obtained using ultrasound-guided percutaneous technique. A pulmonary artery catheter (Edwards Life Sciences, Irvine, California) was inserted via the right external jugular vein. The left external jugular vein was accessed for resuscitation fluids. The right carotid was accessed to monitor blood pressure with a solid-state pressure sensor catheter (Millar Instruments, Houston, Texas) to ensure accuracy of low pressure measurements. The right femoral artery was cannulated with a 14 Fr catheter sheath for blood withdrawal to avoid arterial collapse. Access of the left carotid artery was obtained through invasive cut down to monitor flow rate by a flowprobe (Transonic Systems, Ithaca, New York). Splenectomy was performed through a midline laparotomy to prevent splenic autoperfusion during hemorrhage followed by a cystostomy for urine collection. After weighing, the spleen was returned to the abdominal cavity to regain natural organ geometry. Following abdominal closure, the AAJT was then placed under the animal but not buckled.

Controlled Hemorrhage and Cardiac Arrest

Following a 10-minute stabilization, blood was withdrawn from the right femoral artery using a peristaltic pump (Cole-Parmer, Vernon Hills, Illinois) at a rate of 2 mL/kg/min based on previous studies in our lab.[12,13] This rate was continued until the carotid systolic pressure reached below 10 mm Hg for 10 seconds (defined as arrest, $t = 0$). Hemorrhage was then decreased to 1 mL/kg/min while pulse fluctuations were still visible. After 2 minutes of arrest ($t = 2$), treatment group was determined by opening a sealed envelope with the group identity. If selected, the AAJT was then buckled and inflated per manufacturer's instructions (AAJT), or if not selected, remain unbuckled (CONTROL). CPR was initiated once 3 minutes of arrest had elapsed ($t = 3$) or until the AAJT was fully inflated following the 3 minutes of arrest.

CPR was performed using an oxygen-driven mechanical device (Life-Stat Model 1008, Michigan Instruments, Grand Rapids, Michigan) with compressions set at 5-cm depth and a rate of 100 compressions/minute. Concurrently, fresh whole blood was delivered at 500 mL/min using a Belmont Rapid Infuser (Belmont Instrument, Billerica, Massachusetts) along with calcium chloride at 0.5 g/min. At the onset of compressions, inspired oxygen was increased to 100% for the remainder of the experiment. Ten minutes of compressions were performed before stopping to assess endogenous cardiac rhythm. Compressions were restarted and reassessed every 5 minutes through the 1-hour observation period or until death was called (either no pulse fluctuations or asystole occurred).

Blood for resuscitation was collected into standard blood donation bags containing citrate solution (CPDA-1). Five units total were administered to each animal. Two units were collected during the controlled hemorrhage portion of the protocol and 3 units were collected from a dedicated pool of donor pigs less than 72 hours before use.

Outcomes

The primary outcomes for this study were survival and time of ROSC (systolic >90 mm Hg). Secondary outcomes included physiologic data including hemodynamics, blood chemistries, and tissue oxygenation of the right pectoralis muscle using near infrared spectroscopy with pediatric monitoring

TABLE I. Baseline Values

	n	Weight (kg)	Hemorrhage Time (Minutes)	Blood Loss (g)	SBP (mm Hg)	MAP (mm Hg)	Carotid Blood Flow (mL/min)	Hemoglobin (g/dL)
Control	6	72.2 ± 3.1	17.8 ± 3.1	2610 ± 353	84.2 ± 5.8	62.9 ± 5.7	375 ± 37.7	10.2 ± 0.8
AAJT	6	73.5 ± 6.2	18.7 ± 4.9	2697 ± 317	88.0 ± 10.6	67.5 ± 7.3	394 ± 59.8	10.5 ± 0.5
p Value		0.648	0.728	0.664	0.465	0.255	0.513	0.453

AAJT, abdominal aortic and junctional tourniquet; SBP, systolic blood pressure; MAP, mean arterial pressure.

pads (INVOS 5100C Somatic Oximeter, Medtronic, Minneapolis, Minnesota). The occurrence of ventricle fibrillations during the experiment was considered an exclusion criteria and defibrillation was not included in the protocol.

Data Analysis

Data are presented as mean ± standard deviation, and differences between groups was considered significant when $p < 0.05$. Groups were compared using Student's *t*-test, 2-way repeated measures analysis of variance (ANOVA), or log rank survival analysis as appropriate. All statistical analysis was performed using SigmaPlot 12 (Systat Software, San Jose, California).

RESULTS

Twelve pigs met criteria and were included in the study, but three animals had to be excluded and replaced due to occurrence of ventricle fibrillations. There were no baseline differences between animals before the initiation of hemorrhage (Table I). For all animals included in study, the weight was 72.8 ± 4.7 kg, baseline mean arterial pressure (MAP) was 65.2 ± 6.7 mm Hg, baseline pulse of 70.9 ± 8.6 bpm, and end-tidal CO_2 ($EtCO_2$) was 43.1 ± 1.6 mm Hg.

Controlled Hemorrhage and Arrest

During the controlled hemorrhage to cardiac arrest, the animals behaved as predicted. Each animal underwent a period of tachycardia followed by a decrease in heart rate toward bradycardia and asystole. Blood pressure, $EtCO_2$, mixed venous oxygen saturation, central venous pressure, cardiac output, and near-infrared spectroscopy all decreased towards arrest. There were no significant differences between groups at the time of arrest defined as a systolic pressure of <10 mm Hg (Table II). Overall, hemorrhage took 18.2 ± 3.9 minutes and the withdrawal of 2654 ± 323 g of blood. Compressions were started later in the AAJT group due to longer than anticipated application times, but these differences were not significant (3.1 ± 0.1 vs. 3.4 ± 0.4 minutes, $p = 0.18$).

Survival

Survival time was significantly different between groups as analyzed by log ranks test ($p = 0.02$; Fig. 1). Five of the six animals in the CPR with AAJT group survived to the end of the experiment, whereas only one animal in the CPR alone (CONTROL) group survived to the end. All animals that survived to the end of the experiment had a ROSC during the initial 10-minute compression period. One animal in each group had small pulse fluctuations (<5 mm Hg) following this initial period, but neither achieved a ROSC despite additional chest compressions. Time of ROSC (systolic >90 mm Hg) was 6.9 ± 1.6 minutes for the AAJT group, whereas the one surviving animal from the CONTROL group occurred at 8.0 minutes.

Physiology

Table II shows hemodynamic values of each group from onset of hemorrhage until the end of initial period of compressions. Repeated measures ANOVA showed significant

TABLE II. Arrest and Postcompression Values

	n	SBP (mm Hg)	DBP (mm Hg)	MAP (mm Hg)	Carotid Blood Flow (mL/min)	MPAP (mm Hg)	$EtCO_2$ (mm Hg)	Lactate (mmol/L)	Base (mmol/L)
At Arrest									
Control	6	8.6 ± 1.7	5.2 ± 3.6	6.4 ± 2.9	4.3 ± 4.5	6.9 ± 1.5	7.5 ± 3.9	3.9 ± 0.6	6.2 ± 3.3
AAJT	6	7.8 ± 1.0	2.9 ± 2.5	4.5 ± 1.9	3.8 ± 3.4	7.9 ± 2.9	7.8 ± 2.8	4.0 ± 2.0	4.4 ± 3.6
p value		0.373	0.21	0.227	0.821	0.478	0.87	0.845	0.389
Following 10-Minute Compressions									
Control	6	29 ± 36	24 ± 45	34 ± 52	77 ± 169	21 ± 5.3	21 ± 12	7.0 ± 2.5	−6.2 ± 3.4
AAJT	6	146 ± 49	81 ± 42	100 ± 47	557 ± 357	30 ± 4.4	47 ± 22	5.5 ± 1.5	−3.3 ± 2.2
p value		0.001**	0.048*	0.045*	0.014*	0.010*	0.030*	0.224	0.112

AAJT, abdominal aortic and junctional tourniquet; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; MPAP, mean pulmonary artery pressure; EtCO2, end tidal carbon cioxide. *, $p < 0.05$; **, $p < 0.01$.

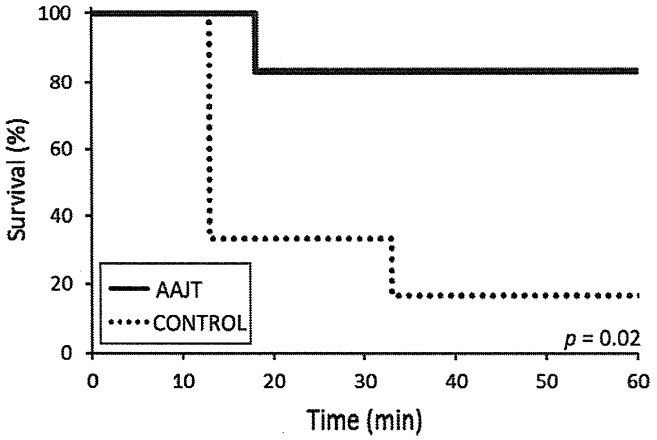

FIGURE 1. Kaplan–Meier analysis of survival time of animals with and without abdominal aortic and junctional tourniquet (AAJT) during cardiopulmonary resuscitation. AAJT animals lived significantly longer than control animals.

differences between groups with respect to carotid systolic pressure ($p = 0.033$). Diastolic arterial pressure, MAP, and carotid flow were not significant by repeated measures ANOVA ($p = 0.076$, 0.052, and 0.085, respectively; Fig. 2). However, a *t*-test performed following the end of compressions showed that carotid systolic, diastolic, and MAPs

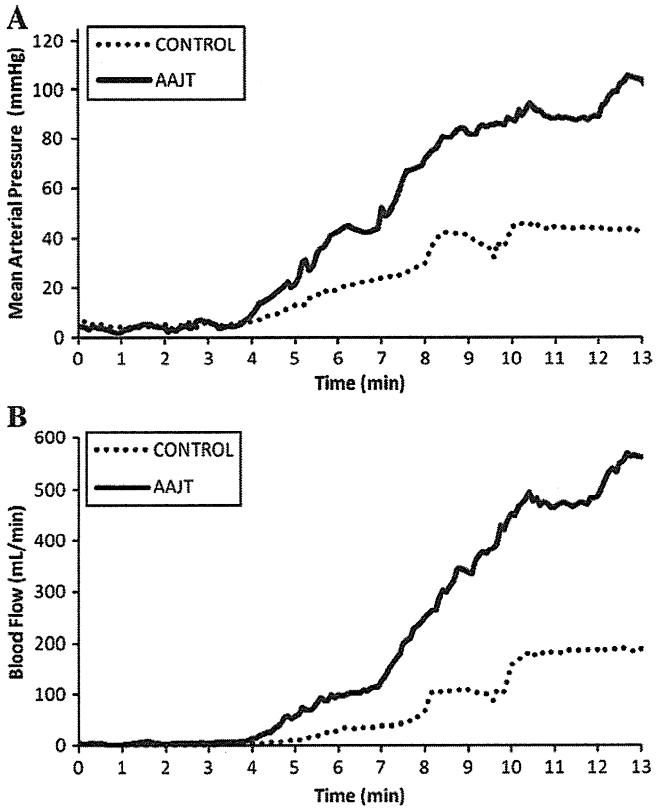

FIGURE 2. Hemodynamic effects following arrest. (A) Blood pressure measurement from time of arrest through the first ten minutes of compression. Differences are not significant ($p = 0.052$). (B) Blood flow measurements over the same period. Differences are not significant ($p = 0.085$)

along with carotid flow, $EtCO_2$, and mean pulmonary artery pressure were all significantly different between groups.

DISCUSSION

This study explored the possibility of using the AAJT as an adjunct to improve CPR efficacy following TCA in a swine model. A controlled hemorrhage was used to reach near cardiac standstill in order to simulate massive hemorrhage leading to cardiac arrest. After 3 minutes of arrest, CPR was performed with or without the tourniquet along with rapid fluid resuscitation. The results presented here indicate that survival and other measures of CPR efficacy were significantly improved at the end of chest compressions with AAJT. Importantly, the AAJT appears to show an increase in survival and ROSC, the end goal of CPR. Therefore, in laboratory conditions with large volumes of fresh whole blood, the AAJT is effective in facilitating CPR.

Standard CPR is believed to be ineffective in cases of TCA, but there has been little published research on the subject. In 1989, Luna and colleagues showed that CPR in hypovolemic animals is ineffective and reduces diastolic blood pressure (a key indicator of the efficacy of delivered CPR) in a baboon model of cardiac arrest.[9] More recently, a study using a canine model of pulseless electrical activity showed that infusion of fluids was more efficient in treating signs of shock than CPR alone.[14]

The mechanism whereby AAJT facilitates CPR efficacy in these experiments is unknown. One possibility is that AAJT application sequesters fluid resuscitation to the area above the tourniquet. This would allow an overall increase in vascular pressure, cardiac preload, and coronary artery pressure compared to the animals without the tourniquet. Another possibility is that the AAJT allows for more effective compressions while in place due to the counterpressure produced by the AAJT on the diaphragm. This possibility is supported by the limited success seen with previous reports involving an abdominal cuff and military anti-shock trousers during CPR.[17] Finally, since the AAJT blocks both arterial and venous blood flow, each compression may force the blood into coronary arteries as well as the carotid and other major vessels without losing energy to the lower extremities. Further research can differentiate between these possible mechanisms that lead to an increased CPR efficacy.

This study suffers from limitations inherent in animal models of trauma and hemorrhage. Although similar, there are anatomical differences between humans and swine. Notably, swine torsos are round, whereas human torso are flatter necessitating the use of the wedge-shaped table during chest compressions. Swine hindlimbs are shorter than human legs and application of the AAJT sequesters less blood in swine than in humans, but it is unclear how these differences would have affected the outcomes measured here. Additionally, the model of hemorrhage-induced TCA used here does not contain trauma beyond that which occurs during line placement and splenectomy. No evaluation of

neurological outcome was performed. Also, this experiment, although being randomized and blinded, had relatively few animals in each group ($n = 6$). Despite these limitations, this study advances our knowledge of possible CPR adjuncts during TCA.

Further study is needed to fully understand the benefit of having the AAJT in place during CPR. Variables including time of arrest and resuscitation volume/rate need to be explored. Elucidating the mechanism whereby AAJT improves CPR in extreme hypovolemia could lead to knowledge or devices that are even more efficient in improving CPR.

CONCLUSIONS

The addition of AAJT improved ROSC in a hemorrhagic cardiac arrest swine model. Further research is needed to determine the applicability of the AAJT to a wider array of injury types and resuscitation paradigms.

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